found that even "relatively mild stress" depressed cellular immunity in healthy adults. Kiecolt-Glaser et al. (1984) found that high scorers on stressful life events and loneliness had significantly lower levels of NK cell activity. Herberman (1988), Irwin, Vale & Britton (1987), Nemeroff et al. (1984) and Rou et al. (1988) found impaired immune reaction in depressed groups, and Linn, Linn & Klimas (1988), Arnetz et al. (1987), Glaser, Kiecolt-Glaser, Speicher & Holliday (1985) and Shavit et al. (1989) found impaired immune reactions to stress. Jemmott and Magloive (1988) found that stress lowered salivary concentrations of S-IgA, while social support increased them. Grossarth-Maticek and Eysenck (1989) found that behaviour therapy significantly increased the percentage lymphocyte count in terminally ill women suffering from cancer, and also increased their survival time. Pennebaker et al. (1988) found that self-disclosure improved cellular immune functioning. Kiecolt-Glaser et al. (1985) found an enhancement of immunocompetence by relaxation and social contact.

Irwin, Daniels, Bloom, Smith and Weiner (1987) have shown that life events can cause depression, and reduce the effectiveness of the immune function. Similarly, Murphy, Monson, Sabol and Leighton (1987), in a prospective study of 1003 adults, found a significant correlation between depression and mortality. Rodin (1980, 1986) showed that appropriate psychotherapy reduced depression and cortisol level through psychotherapy. Dabbs and Hopper (in press) showed that cortisol level correlated with anxiety, depression and high heart rate.

Finally, the relationship between mood and the immune system response has been established in a series of studies (e.g. Baker, 1987; Dillon and Baker, 1985; Linn, Linn & Jensen, 1984; McClelland et

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al., 1989, 1985; Morgan, 1989, and Stone et al., 1987). Animal studies, too, have contributed to the formulation of the model (e.g. Glaser et al., 1985; Landenslager et al., 1983; Borysenko and Borysenko, 1982; and for a review, Justice, 1985.)

The studies quoted are only among the most recent; for reviews of the older and perhaps less convincing material the following are suggested: Jemmett and Locke (1984), Miller (1983, 1985), Baker (1987), Kennedy et al. (1988), Testima (1986), Pletnikoff, Faith, Murgo and Good (1986), Korneva, Klimento and Shkhinek (1985), Antoni (1987) and Steptoe (1989). Taking all the published data together, they do seem to support the sort of model suggested by Dilman and Ostronmova (1984) and Eysenck (1986), and briefly outlined above. There is evidence that (1) personality and stress produce immunodestructive substances in the bloodstream; (2) that these substances do have such an immunodestructive function, and that (3) behavioural manipulations can reverse this process. Thus there appears to exist at least a preliminary model to explain along causal lines the effectiveness of behaviour therapy in prophylaxis for cancer, and in prolonging life in cancer sufferers.

There is one apparent objection to this argument. As Zanderman, Costa and McCrae (1989) have shown, there is no evidence in a nationally representative sample for any correlation between depressive symptoms and cancer morbidity. The answer to this is very simple. Depression is a multi-faceted set of symptoms, like fever, which may have diverse causes and relate to different disorders; the difference between reactive and endogenous depression is perhaps the best known. The type of depression referred to in our theory is sub-clinical, and might be defined as "hopelessness depression" (Alloy, Abramson, Metalsky and Hartlage, 1988). This concept is largely based

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on the work of Seligman (1975) and Abramson, Seligman and Teasdale (1978), and is essentially a cognitive diathesis-stress theory of depression (Alloy, Clements and Holden, 1985). According to this theory, "a proximal sufficient cause of depression is an expectation that highly desired outcomes are unlikely to occur, or that highly aversive outcomes are likely to occur, and that no response in one's repertoire will change the likelihood of occurrence of these outcomes." (Alloy et al., 1988 p.7.) It is in this sense that the term has been used in our research. Other varieties of depression may or may not be relevant, and it is important to note that animal work too has emphasized the importance of differentiating between escapable and inescapable shocks, and the vital contribution of predictability (Miller, 1981.)

As far as coronary heart disease is concerned there is less material to review, but sclerosis is an obvious intermediary. Grossarth-Maticek and Eysenck (in press) have reported a study in which 100 cancer-prone and 92 coronary heart disease-prone probands had the degree of sclerosis in the fundus of the eye measured on a 3-point scale by a leading ophthalmologist; before and after therapy (for a randomly selected 50% of probands in each case) and at similar points of time for probands in the control group. Fig. 7 shows the results. Type 2 probands have significantly higher levels of sclerosis than Type 1 probands, and the therapy group a significantly lower degree of sclerosis; more so for CHD-prone Type 2 than for cancer-prone Type 1 probands. This experiment is in urgent need of replication.

Fig. 7 here.

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It is of course necessary to preserve caution in interpreting these results. As in the case of epidemiology generally, there are no singular causes producing single results. Thus Cobb and Rose (1973) showed that air traffic controllers have a higher incidence of hypertension than controls, and produce the condition at a younger age. This could be interpreted as a direct effect of the greater stress under which air traffic controllers work. However, hypertension is strongly related to alcohol consumption in air traffic controllers (De Franks, Jenkinson & Rose, 1987). It is possible that the drinking may be the important determinant, as alcohol intake is known to raise blood pressure (MacMahn, 1987.). Complications of this kind are the rule in this field, rather than the exception.

Clearly the Grossarth-Maticek and Eysenck data concerning the prophylactic effects of behaviour therapy for cancer and coronary heart disease are equally subject to this caution. We have shown (Grossarth-Maticek, Eysenck, Gallasch, Vetter and Frentzel-Beyme, in press) that after therapy there is a very significant shift from Type 1 or Type 2 behaviour to Type 4 behaviour; there is no change in smoking habits. However, as Pearl (1925) has pointed out, there is a style of life which embraces many different behaviour patterns, and changing one may change many others, one of which, or any combination of which, can carry the burden of changes in cancer-proneness or coronary heart disease-proneness. We are only at the beginning of the scientific study of these complex nomological networks, and should not pretend to a greater or more secure understanding of these causal effects than is reasonable under the circumstances.

Eysenck (1989) has discussed the social implications of this type of work. Clearly preventive medicine is much more humane in its consequences, as well as much cheaper, than traditional medicine which

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waits until the disease has manifested itself before attempting any intervention. Many practical, social and ethical problems are raised when we consider the possibility of introducing methods of this kind for the prevention of cancer and coronary heart disease into the social systems at present concerned with health, but it is time that the issues were taken seriously, and debated in a meaningful fashion. These issues are too serious to be left to epidemiologists.

What is most important from the point of view of this book, of course, is the demonstration of the heightened probability of a causal relation between stressed personality, on the one hand, and cancer and coronary heart disease, on the other. The failure to demonstrate such a relationship between smoking and disease, shown in previous sections, is the most vulnerable point in the orthodox view. These results show that it is possible to demonstrate causal relations following accepted scientific methodology, and to establish personality and stress as important risk factors in cancer and coronary heart disease.

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(8) Summary and Conclusions.

In spite of all the criticisms that may justifiably be made of modern epidemiological methods and findings, it is possible to make some fairly definitive statements which characterize the present status of research in the field covered by this book.

(1) It is clear beyond doubt that there is a debate going on concerning risk factors in cancer and coronary heart disease, particularly smoking. On the one hand we have a large group of physicians, epidemiologists and others who claim that smoking is the major cause of cancer and coronary heart disease, and that if only we could stop people smoking large numbers of lives could be saved. On the other hand we have a large number of physicians, epidemiologists, oncologists and other scientists who believe that what we are dealing with is a multiplicity of risk factors, interacting in complex ways, of which smoking is only one, and not by any means the most important. The Press has almost entirely suppressed representation of this alternative view, and has given rise to the completely unjustified and unscientific belief that smoking is the main and almost the only risk factor for cancer and coronary heart disease. It seems about time that the truth regarding the existence of this debate was publicly acknowledged, and that the irresponsible exaggerations concerning the effects of smoking ceased to be published and broadcast.

(2) There is now little doubt that smoking has certain positive effects which account for its popularity, and the curious fact that even people who believe that smoking causes disease still continue to smoke. Among the positive features of smoking which have now been firmly established by research are its ability to raise the cortical

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arousal level, and thus counteract boredom and fatigue; its ability to make anxious and tense persons relax, thus reducing the impact of negative emotions; and its power to increase attention and vigilance, thus enabling people to work longer and harder, and more efficiently, than they could do otherwise. These are important advantages, which should be set against the disadvantages of smoking. There does not seem to be any truth in the allegation that smoking is an addiction, even if that term could be defined in such a way as to have a scientific meaning. People maintain the smoking habit because of the positive effects of smoking on their psychological wellbeing; if we substitute alternative behaviours which serve the same needs, smoking can readily be given up as being no longer required.

(3) The relative importance of smoking as a risk factor, compared with psychosocial and other risk factors, has received a good deal of attention. Roughly speaking it would appear that personality and stress are 6 times as important as smoking in the statistical correlation between disease and risk factors. This does not mean that smoking is necessarily harmless, but it certainly puts into perspective its possible influence on cancer and coronary heart disease.

(4) What emerges with particular clarity from all the work that has been summarized in these pages is the importance of psychosocial factors, i.e. stress and the individual's reaction to stress. Of all the risk factors considered, this is perhaps the most important, showing the strongest correlation with death from cancer and coronary heart disease. It is curious, considering the long history of theories concerning psychosocial factors, going back for over 2,000 years, that the medical profession has on the whole failed to pay much attention to the importance of stress and personality in the causation

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of disease, and tends to reject claims that stress is a real killer. The evidence is too strong now to reject such claims out of hand.

(5) The only model which is remotely adequate to contain the known facts in this field is a multifactorial synergistic model, i.e. one in which many risk factors are included, and where these risk factors act in a synergistic, (i.e. multiplicative), rather than in an additive fashion. Thus in our own work smoking has always been found to be uncorrelated with cancer or with coronary heart disease in groups where there was no stress, or where a healthy personality counteracted the stress and managed to cope with it. Positive correlations with disease do appear in populations of Type 1 or 2, i.e. populations where stress is endemic, and coping behaviour is inadequate.

(6) So far we have discussed only correlations between risk factors like smoking, stress, personality, etc., and disease; it is well known that correlation does not imply causation, and that statistical association may suggest but does not prove causation. The best demonstration that a causal model may be indicated is furnished by intervention studies. If we believe that smoking causes cancer and coronary heart disease, then clearly quitting smoking should reduce the risk of cancer and coronary heart disease. As we have shown, the evidence does not suggest that this is so. Where quitters are self-selected, they already differ profoundly in their state of health from continuing smokers, and any differences in outcome are more likely to be due to this difference than to the effects of quitting. Where quitters are part of a therapy group, their future health does not, or only slightly, differ from that of continuing smokers. The evidence is confused at best, but it certainly does not support the wildly excessive claim that has been

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made for the beneficial effects of quitting smoking.

(7) The position is quite different with respect to psychosocial factors, where the evidence now seems conclusive that the application of the methods of behaviour therapy to people suffering from stress and inadequate coping mechanisms can prevent cancer and coronary heart disease, when groups receiving such help are compared with groups not receiving it. Thus not only are psychosocial factors statistically much more closely related to disease, but there is evidence for a causal influence here which is absent in the case of smoking.

(8) Clearly there must be intermediary factors intervening between stress and personality, on the one hand, and disease, on the other, and the discovery of such factors is one of the major tasks for future research. It seems reasonable to assume that the major intermediary in the case of cancer is the immune system, and there is evidence that behaviour therapy can improve the efficacy of the immune system, while stress has the opposite effect (Eysenck, 1986; Rodin, 1984, 1986). Similarly, there is evidence for a marked influence of stress and behaviour therapy on the degree of sclerosis shown by individual probands (Grossarth-Maticek et al., in press). With this whole field of research being so young, this area has not been sufficiently researched to come to any very definite conclusion, but these are suggestions which are certainly worth following up.

It would not be right to pretend that the position in which we find ourselves is a very comfortable one, or that our knowledge is anything like as adequate to the challenges of disease as it ought to be. One of the major reasons for this unsatisfactory position is that research money has gone into areas which have proved unrewarding.

We may with advantage remind ourselves that the Multiple Risk

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Factor Intervention Trial Study was stated to have cost 115 million dollars, with the true cost being probably appreciably higher; yet that study was fundamentally flawed from the beginning, was badly executed and analysed, and gave results that were embarrassing to the medical profession as a whole. Had the money been spent on proper scientific research we would be very much more knowledgeable than we are now.

In particular, research is vital in the area we know least about, namely the effects of stress and of behaviour therapy on the immune system; prospective studies of the kind pioneered by Grossarth-Maticek (see references) should be repeated with the inclusion of direct measures of immune system assays, and with multiple measures of sclerosis. Only in this way will we achieve greater knowledge of the causes of cancer and coronary heart disease, and of the most efficient methods of preventing them.

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(9) Epilogue.

The question of risk factors for cancer and coronary heart disease, and the relative importance of smoking as a risk factor, is clearly an important scientific problem which should be solved by the use of traditional scientific methods. Yet we have found that conclusions have often been based on inadequate methodology, doubtful assumptions, inappropriate statistics, and inadmissible extrapolations. It seems that evangelical fervour, dogmatic certitude and propagandist intentions have only too often led to a premature crystallization of spurious orthodoxies which has set back the factual study of the subject and delayed the finding of proper answers by many years. It is unfortunate that the issue has became clouded by an anti-smoking crusade which pays scant regard to the facts of the case, but uses facts selectively to prove a conclusion already predetermined on a priori grounds.

In this the whole controversy resembles similar controversies which have ranged around issues like the role of cholesterol in CHD, or that of fluoridation of the water supply. In both cases, publicity campaigns were waged in order to get people to substitute margarine for butter, change the whole diet to lower cholesterol levels, increase the fluoride level of the water supply, and quite generally change people's habits in line with supposititious benefits to their health. Had these benefits been real, and had these changes in fact produced better health and longer life, the fanatical support given these measures might be understandable. However, as already noted, there do not seem to be any obvious benefits to be derived from a

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cholesterol-low diet, and in fact it might be linked with increasing risks of cancer. Similarly, fluoride in the water supply is now stated to provide little if any benefit as far as tooth decay is concerned and may indeed have the opposite effect. Furthermore, it has been suggested that it may be linked with increasing risks of cancer.

I cannot pronounce on these developments as far as their medical background is concerned; the new developments may have as little substance as the old. They may, however, have the beneficial effect of making lay readers, and possibly even medical readers, less prone to take seriously any pronouncements of epidemiological "experts" based on insufficient evidence and grossly defective studies. What is being criticized is not the individual scientist who draws the wrong conclusion from his data, or who commits errors of one kind or another in his experimental methodology, or his statistical analysis. To err is human, and all scientists are prone to blunders of one kind or another.

But scientists should be modest in their claims, should refuse to exaggerate the certainty of their conclusions, and should always be open to correction and the consideration of contrary evidence. In particular, they should refrain from advocating far-reaching social actions on the basis of doubtful, fallible, disputed and insufficient evidence. Equally, they should not pretend that contradictory data do not exist, that anyone who doubted the correctness of their arguments was acting on improper motives, or that it was irresponsible to argue against their firm convictions. Yet the propounders of the low cholesterol diet, and the advocates of fluoridation, have been guilty of all these misdemeanours, as have those who have condemned cigarette-smoking out of hand as being responsible for the majority of

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deaths from cancer and CHD.

Here, as with cholesterol and fluoride, scientists have been guilty of suppressio veri and suggestio falsi, of neglecting to inform the public of facts contrary to their beliefs, of public statements going way beyond the established facts, of emotional propaganda not based on solid evidence, and altogether of fanatical publicity-seeking quite alien to the received model of the genuine scientist. There is no culpability in being wrong, but there is culpability in perpetuating error in the face of contradictory evidence, in bigoted and zealous over-enthusiasm, and in prematurely urging major social intervention on the basis of doubtful and contradictory evidence.

The grossly exaggerated claims concerning the degree of risks attending cigarette smoking in the causation of cancer and CHD must stand with the grossly exaggerated claims concerning cholesterol and fluoride as a warning to scientists to avoid jumping on bandwagons, join publicity campaigns, or advocate premature social action. As Claude Bernard warned a hundred years ago: "In ignorance, abstain!"

There is one additional matter that requires discussion in this context. The premature claims for the preventive possibilities of a reduced cholesterol diet in relation to CHD, fluoridation in relation to tooth decay, and giving up smoking in relation to cancer and CHD, and the widespread publicity these have received through the media, have suggested to the layman that a far greater certitude attends to these claims than can be justified scientifically, as we have seen. The recent findings that these claims are in fact unfounded, and that fluoridation, or indulgence in a low cholesterol diet may in fact harm rather than benefit the individual, have led to bewilderment, and a feeling, only too well justified, that "expert" opinion on medical

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matters is quite untrustworthy, and that the constant reversals of recommendations for a healthier living emanating from such sources indicate a lack of true knowledge, and a partisanship unworthy of scientists. As Le Fanu (1987) has pointed out, in the 30s we were advised by nutritionists to eat more eggs and butter, and drink more milk. A few years later all these nutriments became anathema, in the interests of a low cholesterol diet. Now we are being told that this was all a mistake, and that there is no harm in eggs, butter and milk, and that they may even be good for us. Clearly something has gone wrong very badly when violently opposite recommendations are made on the apparent basis of "scientific" evidence, and the public has a right to be bewildered, and to doubt if such advice is really based on fact rather than passing fancy. This undermines public faith in the objectivity of science, and the truth-value of scientists' published beliefs; it is, indeed, the old story of the boy who cried 'Wolf'. Having cried wolf so often, only to retract the statements of the alleged danger, nutritionists, epidemiologists and other medical specialists are now in danger of being disbelieved even when their warnings are only too well justified.

Worse yet, warnings which are not justified by the state of our present knowledge may themselves increase stress, and lead to very serious health consequences. Our study (Grossarth-Maticek & Eysenck, 1989) has shown that such fears are indeed only too real, and that the constantly repeated media threat that smoking kills has in fact become a self-fulfilling prophecy! Thus the lack of responsibility shown in making publicly unjustified statements about the evil consequences of smoking is underlined by the actual consequences of making such statements, namely the deaths of numerous people frightened literally out of their lives by the media and their grossly and very partial

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accounts of the evidence (Clark, 1989). When the assertions made by apparently authoritative bodies and persons, such as the Royal College of Physicians or the Surgeon-General of the United States, grotesquely exaggerate the consequences of smoking in deaths from cancer and CHD, and the virtues of giving up smoking, it is small wonder that the media, as well as politicians, take up the chorus, not realizing that there is little justification for the claims made, and that they have received damaging criticism from leading oncologists, immunologists, and other scientists.

The only responsible way out of this practice, which is seriously embarrassing for medicine and science alike, is to return to the precepts which have always guided science in the past. Publications should contain not only assertions of position, based on selected and biased evidence, but should deal fairly and squarely with all objections, criticisms and contrary findings. Conclusions should be justified by the evidence, and carefully qualified rather than extreme in their certainty when this is not warranted. Finally, no publicity should be given to results, and no publication urged or taken, unless there is virtual agreement among experts (as there most certainly is not in relation to the effects of smoking) about the issue in question. Even then the fallability of research in such very complex areas should be emphasized, and the possibility of error acknowledged. These are the ways science has progressed, and has acquired its unique prestige; we should be careful not to squander it in the way that it has been squandered in relation to these controversies.

What is particularly to be regretted is the intrusion of political associations into scientific debates. It is interesting to note that one of the most determined protagonists of the "smoking causes disease" view, (Everett Koop, the former U.S. Surgeon-General)

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has recently been condemned by a House of Representative Committee for suppressing evidence on another issue, also involving social and ethical considerations, namely abortion. According to the Committee, Koop refused to publish a study of the physical and psychological effects of abortion on women, because the study found no evidence that the procedure harms women. At least one scientist working at the U.S. Government's Center for Disease Control, whose research focused on abortion, had his findings censored, and another was demoted, according to the Committee. Apparently when the Report commissioned by the White House had failed to provide the hoped-for evidence condemning abortion, "he therefore decided not to issue a report, but instead to write a letter to the president which would be sufficiently vague as to avoid supporting the pro-choice position that abortion is safe for women." Federal funding of research on contraception had halved since 1980, Government officials often treated those who did research on abortion with hostility, and officials who did not obey the anti-abortion line and dared to contradict Koop were fired or transferred, and their writing censored.

These are precisely the tactics that have been used widely in connection with the "smoking causes disease" issue. It has become difficult for those who wish to examine the problem objectively to obtain research funds, or to publish their data, if they are not in line with official policy. Newspapers refuse to discuss the facts objectively, and pretend that unanimity exists when in reality confusion reigns, and criticism of the "orthodox" view is widespread. Alternative models are dismissed without proper examination, and are never mentioned in official publications. Investigators who show an interest in such models encounter obstacles in their careers, and may have all support withdrawn, regardless of the quality of their

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research. These are not conditions which encourage high-class scientific research, and it is small wonder that the field is confused and full of anomalies. In this book I have tried to set out the facts as carefully and objectively as possible, and to point out the major conclusions which may be drawn.

The failure of many workers in this field to be objective, and to tell the public fairly and squarely about the present position of research, has been exposed most convincingly by Effron (1984) in her book on "The Apocalyptics", which cuts a much wider swathe than the present book, but which justifies amply its subtitle: "Cancer and the big lie". It deals with "the ideological corruption of cancer research in the United States." The author contends, and demonstrates, that an extreme environmental movement, which she calls "the apocalyptics", has politically distorted research in environmental cancer, saturating the United States with theories of cancer that are pure myths. Her book should be read by all concerned with the problem of cancer origin and propagation; the problem considered in this book is only one facet of this much wider phenomenon. There is no doubt that smoking is one of many risk factors in cancer and CHD, but the notion that it is all-important, and that reduction in smoking would have far-reaching benevolent consequences as far as cancer and CHD are concerned, is just one of those myths which Effron castigates.

There is much that remains obscure, but there are also some features of the scene which must be regarded as hopeful. If stress and other psychosocial factors are indeed killers, it does seem that suitable prophylactic means are at hand to delay or prevent cancer and coronary heart disease. Behaviour therapy is a very cheap and convenient way of safeguarding healthy probands from cancer and

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coronary heart disease, and from every point of view prevention must be better than largely non-existent cures or very expensive treatments with often disastrous side-effects. If only we could disabuse ourselves of the present unhealthy preoccupation with smoking, and turn our attention to the real enemy, namely stress, we could in reality save all those lives which a dishonest propaganda effort has tried to persuade us could be saved if only we gave up smoking. This is a serious issue, vital for hundreds of thousands of people, and it is irresponsible to continue a bland and biased disregard of the true facts of the case.

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# CAPTIONS

Table 1. Comparison of CHD morbidity ratios and incidence rates of never-smokers and ex-smokers at various years of follow-up. (After Seltzer, 1989.)

" 2. CHD risk ratios (cigarette smokers/non-smokers) calculated from the Framingham Heart Study incidence data. (After Seltzer, 1989.)

" 3. Comparison of U.S. Surgeon-General's coronary heart disease findings and those of the Framingham Study data with respect to cigarette smoking. (After Seltzer, 1989.)

" 4. Causes of death for different types of probands - Yugoslav sample. (Grossarth-Maticek & Eysenck, 1980.)

" 5. Causes of death for different types of probands - Heidelberg normal sample. (Grossarth-Maticek & Eysenck, 1988.)

" 6. Causes of death for different types of probands - Heidelberg stressed sample. (Grossarth-Maticek & Eysenck, 1988.)

" 7. Interaction between smoking, personality and death from lung cancer. (Grossarth-Maticek & Eysenck, 1988.)

" 8. Relative importance of personality and organic risk factors for death. (Grossarth-Maticek, Eysenck & Vetter, 1988.)

" 9. Synergistic effects of H (heredity), B (chronic bronchitis), C (cigarette smoking) and S (stress/personality), BT (behaviour therapy.) (Grossarth-Maticek & Eysenck, in press.)

" 10. Effects of behaviour therapy on cancer and coronary heart disease. (Grossarth-Maticek & Eysenck, 1989.)

" 11. Effects of group behaviour therapy on cancer and coronary heart disease. (Grossarth-Maticek & Eysenck, 1989.)

" 12. Effects of bibliotherapy and short behaviour therapy on cancer and coronary heart disease. (Grossarth-Maticek & Eysenck, 1989.)

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TABLE 1.

| Follow-up. | Morbidity ratios* or incidence rate | | Proportionate difference |
|---|---|---|---|
| 9.1 yr | Never smokers | 77 | +67% |
| | Past " | 46 | |
| 12-yr ("heart attacks") | Never smokers | 82 | +39% |
| | Ex-smokers | 59 | |
| 16-yr | Never smokers | 91 | +49% |
| | Ex-smokers | 61 | |
| 18-yr ("heart attacks") | Never smokers | 17.7~ | +36% |
| | Ex-smokers | 13.0~ | |
| 22-yr | Never smokers | 29.8~ | +23% |
| | Ex-smokers | 24.2~ | |

*Ratio of observed to expected rate (100 times those observed divided by those expected).
~CHD incidence rates per 100 men.

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TABLE 2

| Follow-up | Incidence | Ages | Nonsmoker classification for for comparison with smokers^ | Risk Ratios Men | Women |
|---|---|---|---|---|---|
| 9.1 yr | After Exam 1 | 40-59 | Never smokers | 1.5~ | - |
| | | | Nonsmokers (never, past, <half day) | 1.8~ | - |
| 12-yr | After Exam 1 | 30-59 | Non-cigarette | 1.8\| | 1.0\| |
| 14-yr | After Exam 1 | 35-64 | None | 1.4 | 0.9 |
| 16-yr | After Exam 1 | 35-64 | None | 1.3 | 0.9 |
| | Average annual incidence rate | 35-74 | None | 1.3 | 0.8 |
| 18-yr | Average annual incidence rate | 45-74 | None | 1.3 | 1.0 |
| 20-yr | Average annual "smoothed" rate | 45-74 | None | 1.4 | - |
| 22-yr | After Exam 1 | 30-59 | Non-cigarette | 1.3 | 0.8 |
| 24-yr | After Exam 1 | 30-59 | Non-cigarette | 1.3 | 0.9 |
| 26-yr | After Exam 1 | 35-84 | Non-cigarette | 1.3@ | - |
| 30-yr | Average annual incidence rate | 35-64 | None | 1.6 | 1.1 |
| | | 35-74 | | 1.3 | 1.0 |
| | | 35-84 | | 1.0 | 1.0 |

* Age-adjusted by assigning equal weights to rates in each age-group, in accordaince with Framingham method listed in Table 1.

^ Framingham categories of "none" and "non-cigarette" include ex-smokers, and pipe and cigar smokers in addition to "never smokers".

~ "Standardized incidence ratios" with CHD limited to non-fatal and fatal MI, and sudden CHD death.

| Framingham "morbidity ratios".

@ Framingham "coronary morbidity".

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TABLE 3

| Criteria | U.S. Surgeon-General's findings | Framingham data findings |
|---|---|---|
| Men-Univariate association | Strong | Weak |
| Men-Multivariate " | Strong | Non-significant; absent |
| Women-Univariate " | Present, but less than in men | Absent |
| Women-Multivariate " | Present | Absent |
| Duration of cigarette smoking | Increase of CHD with increase of duration of smoking | Absent |
| Cessation of cigarette smoking | Gradual reduction of CHD to level of non-smokers | Immediate reduction of CHD to level below that of never smokers. |

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Table 4.

| Yugoslavia | Living | Cancer | Coronary heart disease | Other causes of deaths | Total *N* |
|---|---|---|---|---|---|
| Type 1 | 72 = 23.8% | 140 = 46.2% | 25 = 8.3% | 66 = 21.8% | 303 |
| Type 2 | 96 = 28.3% | 19 = 5.6% | 99 = 29.2% | 125 = 36.9% | 339 |
| Type 3 | 123 = 56.7% | 4 = 1.8% | 20 = 9.2% | 70 = 32.3% | 217 |
| Type 4 | 437 = 90.7% | 3 = 0.6% | 8 = 1.7% | 34 = 7.1% | 482 |
| Impossible to allocate to type | 6 | 0 | 4 | 2 | 12 |
| Total | 734 = 54.2% | 166 = 12.3% | 156 = 11.5% | 297 = 27.0% | 1353 |

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Table 5.

| Heidelberg normal | Living | Cancer | Coronary heart disease | Other causes of deaths | Total *N* |
|---|---|---|---|---|---|
| Type 1 | 78 = 71.6% | 19 = 17.4% | 2 = 1.8% | 10 = 9.2% | 109 |
| Type 2 | 109 = 64.1% | 10 = 5.9% | 23 = 13.5% | 28 = 16.5% | 170 |
| Type 3 | 185 = 98.4% | 0 | 1 = 0.5% | 2 = 1.1% | 188 |
| Type 4 | 387 = 99.0% | 0 | 1 = 0.3% | 3 = 0.8% | 391 |
| Impossible to allocate to type | 14 | 0 | 0 | 0 | 14 |
| Total | 773 = 88.6% | 29 = 3.3% | 27 = 3.1% | 43 = 4.9% | 872 |

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Table 6.

| Heidelberg 'stressed' group | Living | Cancer | Coronary heart disease | Other causes of death | Total N |
|---|---|---|---|---|---|
| Type 1 | 188 = 38.4% | 188 = 38.4% | 34 = 7.0% | 79 = 16.2% | 489 |
| Type 2 | 148 = 47.9% | 7 = 2.3% | 86 = 27.8% | 68 = 22.0% | 309 |
| Type 3 | 153 = 92.7% | 4 = 2.4% | 0 | 8 = 4.8% | 165 |
| Type 4 | 71 = 97.3% | 0 | 0 | 2 = 2.7% | 73 |
| Impossible to allocate to type | 6 | 0 | 0 | 0 | 6 |
| Total | 566 = 54.3% | 199 = 19.1% | 120 = 11.5% | 157 = 15.1% | 1042 |

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Table 7.

| | | Yugoslavia | | | Heidelberg (stressed) | | |
|---|---|---|---|---|---|---|---|
| | | Lung cancer deaths | Other deaths | Total | Lung cancer deaths | Other deaths | Total |
| Non-smokers | Type I | 1 = 0.8% | 118 | 119 | 9 = 3.8% | 227 | 236 |
| | Others | 0 | 550 | 550 | 3 = 1.0% | 297 | 300 |
| Smokers | Type I | 31 = 16.9% | 153 | 184 | 37 = 14.6% | 216 | 253 |
| | Others | 6 = 1.2% | 482 | 488 | 0 | 247 | 247 |

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TABLE 8.

| | Type | Mean Y | Mean H1 | Mean H2 | b Y | b H1 | b H2 | Mort. (per cent) Y | Mort. (per cent) H1 | Mort. (per cent) H2 |
|---|---|---|---|---|---|---|---|---|---|---|
| rf: systolic blood pressure | 1 | 151.0 | — | 174.2 | 0.056 | — | 0.024 | 7.6 | — | 7.2 |
| dis: infarct/stroke mortality | 2 | 160.7 | — | 207.6 | 0.084 | — | 0.108 | 27.2 | — | 23.7 |
| | 3 | 148.3 | — | 186.3 | 0.005 | — | 0.010 | 7.7 | — | 2.3 |
| | 4 | 144.6 | — | 185.8 | 0.003 | — | 0.021 | 1.8 | — | 5.0 |
| | all | 150.7 | — | 187.7 | 0.035 | — | 0.041 | 11.1 | — | 9.1 |
| Significance of differences | | 0.0000 | — | 0.0000 | 0.0004 | — | 0.0011 | <0.0001 | — | <0.0001 |
| rf : diastolic blood pressure | 1 | 90.0 | — | 85.8 | 0.026 | — | 0.019 | 5.1 | — | 7.2 |
| dis.: infarct/stroke mortality | 2 | 93.7 | — | 93.5 | 0.093 | — | 0.071 | 26.8 | — | 24.5 |
| | 3 | 88.6 | — | 88.9 | 0.020 | — | 0.004 | 8.5 | — | 2.5 |
| | 4 | 86.8 | — | 89.0 | 0.014 | — | 0.007 | 2.1 | — | 5.5 |
| | all | 89.6 | — | 88.8 | 0.038 | — | 0.025 | 10.6 | — | 9.9 |
| Significance of differences | | 0.0000 | — | 0.0000 | 0.0137 | — | 0.0494 | <0.0001 | — | <0.0001 |
| rf: blood cholesterol | 1 | 255.6 | 217.5 | 258.3 | 0.011 | 0.004 | 0.037 | 8.3 | 3.2 | 8.5 |
| dis.: infarct/stroke mortality | 2 | 250.4 | 254.4 | 305.1 | 0.036 | 0.054 | 0.046 | 29.4 | 9.7 | 24.7 |
| | 3 | 245.8 | 216.8 | 282.6 | 0.027 | 0.000 | 0.003 | 9.2 | 1.8 | 1.8 |
| | 4 | 245.5 | 217.9 | 280.5 | 0.001 | 0.001 | 0.000 | 1.8 | 0.8 | 3.5 |
| | all | 249.1 | 224.9 | 277.8 | 0.019 | 0.014 | 0.020 | 12.2 | 3.9 | 9.6 |
| Significance of differences | | 0.0330 | 0.0000 | 0.0000 | NS | NS | 0.0486 | <0.0001 | 0.0008 | 0.0000 |
| rf: cigarettes per day | 1 | 15.7 | 13.0 | 16.9 | 0.000 | 0.003 | 0.000 | 7.7 | 4.1 | 6.9 |
| dis.: infarct/stroke mortality | 2 | 10.4 | 18.8 | 14.4 | 0.038 | 0.050 | 0.161 | 29.8 | 12.0 | 27.6 |
| | 3 | 11.6 | 8.9 | 12.2 | 0.021 | 0.016 | 0.007 | 8.3 | 2.5 | 1.7 |
| | 4 | 10.8 | 8.2 | 10.9 | 0.002 | 0.008 | 0.011 | 1.8 | 1.2 | 3.5 |
| | all | 11.9 | 11.1 | 15.0 | 0.005 | 0.019 | 0.045 | 11.9 | 5.0 | 9.9 |
| Significance of differences | | 0.0000 | 0.0000 | 0.0000 | NS | NS | 0.0000 | <0.0001 | 0.0002 | 0.0001 |
| rf: cigarettes per day | 1 | 15.7 | 13.0 | 16.9 | 0.075 | 0.019 | 0.044 | 8.2 | 3.6 | 8.3 |
| dis.: lung cancer mortality | 2 | 10.4 | 18.8 | 14.4 | 0.020 | 0.010 | 0.003 | 2.5 | 2.5 | 1.5 |
| | 3 | 11.6 | 8.9 | 12.2 | 0.003 | 0.015 | 0.007 | 1.3 | 2.4 | 1.7 |
| | 4 | 10.8 | 8.2 | 10.9 | 0.002 | 0.008 | 0.011 | 0.6 | 1.2 | 3.5 |
| | all | 11.9 | 11.1 | 15.0 | 0.025 | 0.013 | 0.016 | 3.1 | 2.4 | 3.7 |
| Significance of differences | | 0.0000 | 0.0000 | 0.0000 | 0.0001 | NS | 0.0113 | <0.0001 | NS | 0.0000 |

largest (abs.) value is underlined

For Yugoslavia, the organic variables represent a single measurement taken in 1966 (cholesterol: 1969).
For Heidelberg, the organic variables are the average of up to 7 measurements taken in 1972.
Abbreviations:

rf = risk factor.
dis. = disease.
mean = mean of organic variable within type groups.
b = regression coefficient of the dependent variable on the organic variable within type groups.
mort. = mortality (the dependent variable) within type groups, adjusted for the organic variable.
Y = Yugoslavia H1 = Heidelberg representative H2 = Heidelberg stressed. NS means $P > 0.05$.

20255025231

Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

Table 9.

| Combination of risks: | N | Lung cancer | % | Other causes of death | % | Average Age |
|---|---|---|---|---|---|---|
| Only H | 50 | 0 | 0 | 5 | 10 | 51 |
| Only C | 100 | 0 | 0 | 12 | 12 | 52 |
| Only S | 59 | 0 | 0 | 16 | 27 | 52 |
| H + C | 50 | 1 | 2 | 4 | 8 | 53 |
| H + B | 52 | 0 | 0 | 8 | 15 | 51 |
| C + B | 55 | 0 | 0 | 11 | 20 | 52 |
| C + S | 100 | 2 | 2 | 21 | 21 | 53 |
| H + S | 49 | 0 | 0 | 9 | 18 | 54 |
| B + S | 50 | 0 | 0 | 8 | 16 | 53 |
| C + H + B | 26 | 2 | 8 | 5 | 19 | 51 |
| C + H + S | 50 | 10 | 20 | 14 | 28 | 51 |
| C + B + S | 51 | 5 | 10 | 10 | 20 | 51 |
| H + C + B + S | 26 | 8 | 31 | 8 | 31 | 52 |
| (H+C+B+S+BT) | 26 | 3 | 12 | 4 | 15 | 52 |

2025502532

Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

Table 10.

| I. | N | CANCER Deaths | CANCER Incidence | Other causes of death | Living |
|---|---|---|---|---|---|
| Control | 50 | 16<br>32% | 21<br>42% | 15<br>30% | 19<br>38% |
| Therapy | 50 | 0<br>0 | 13<br>26% | 5<br>10% | 45<br>90% |
| TOTAL: | 100 | 16<br>16% | 34<br>34% | 20<br>20% | 64<br>64% |

| II. | N | CHD Deaths | CHD Incidence | Other causes of death | Living |
|---|---|---|---|---|---|
| Control | 46 | 16<br>34.8% | 20<br>43.5% | 13<br>28.3% | 17<br>36.9% |
| Therapy | 46 | 3<br>6.5% | 11<br>23.9% | 6<br>13% | 37<br>80.4% |
| TOTAL: | 92 | 19<br>20.6% | 31<br>33.7% | 19<br>20.7% | 54<br>58.7% |

2025502533

Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

Table 11.

| | Therapy | | Control | |
|---|---|---|---|---|
| N | 245 | | 245 | |
| Not contacted | 6 | | 11 | |
| | Mortality | Incidence | Mortality | Incidence |
| | 239 | 235 | 234 | 231 |
| Cancer | 18<br>7.5% | 75<br>31.9% | 111<br>47.4% | 129<br>55.8% |
| CHD | 10<br>4.2% | 29<br>12.3% | 36<br>15.4% | 45<br>19.5% |
| Other causes of death | 20<br>8.4% | - | 33<br>14.1% | - |
| Living | 191<br>79.9% | | 56<br>23.9% | |

2025502534

Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

Table 12.

| | Causes of Death | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Cancer | | CHD | | Other | | Total | Living | Not investigated | |
| | D* | I* | D* | I* | D* | I* | | | | |
| Control N=500 | 106<br>21.5% | 162<br>33.4% | 145<br>29.4% | 203<br>41.8% | 164<br>33.3% | – | 415<br>84.2% | 78<br>15.8% | 7<br>1.4% | 15<br>3% |
| Control with use of psycho-analytic text. (Placebo Group.) N=100 | 22<br>22% | 37<br>37.7% | 31<br>31% | 40<br>40.8% | 28<br>28% | – | 81<br>81% | 19<br>19% | 0<br>0% | 2<br>2% |
| Therapy Group with Behaviour Therapy Text. N=600 | 27<br>4.5% | 99<br>16.9% | 47<br>7.9% | 132<br>22.5% | 115<br>19.2% | – | 189<br>31.6% | 409<br>68.4% | 2<br>0.3% | 14<br>2.3% |

* D = died; I = incidence

2025502535

Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

Fig. 1. Cumulative experience of intercourse by age of first intercourse for college educated males and females in the USA. (Data from the National Longitudinal Study.)

" 2. Effect of different types of treatment on giving up smoking. (O'Connor & Stravynski, 1982.)

" 3. Personality type and cause of death, Yugoslav study. (Grossarth-Maticek & Eysenck, 1988.)

" 4. Personality type and cause of death, Heidelberg normal sample. (Grossarth-Maticek & Eysenck, 1988.)

" 5. Personality type and cause of death, Heidelberg stressed sample. (Grossarth-Maticek & Eysenck, 1988.)

" 6. Synergistic action of 7 risk factors for cancer. (Grossarth-Maticek & Eysenck, in press a.)

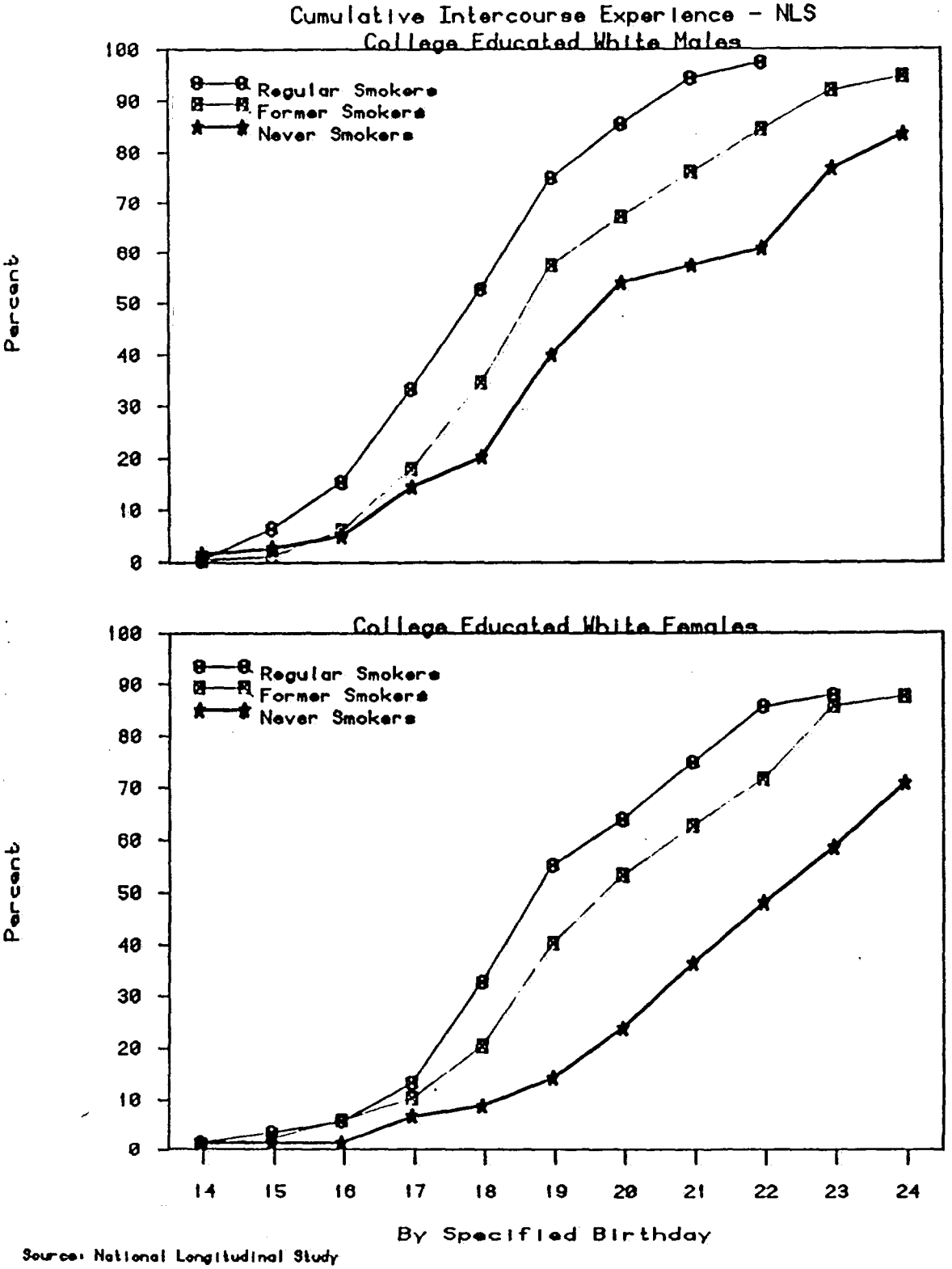
" 7. Sclerosis in cancer-prone and coronary heart disease-prone probands and effects of behaviour therapy. (Grossarth-Maticek & Eysenck, in press b.)



2025502536

Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

FIG. 1




Source: National Longitudinal Study

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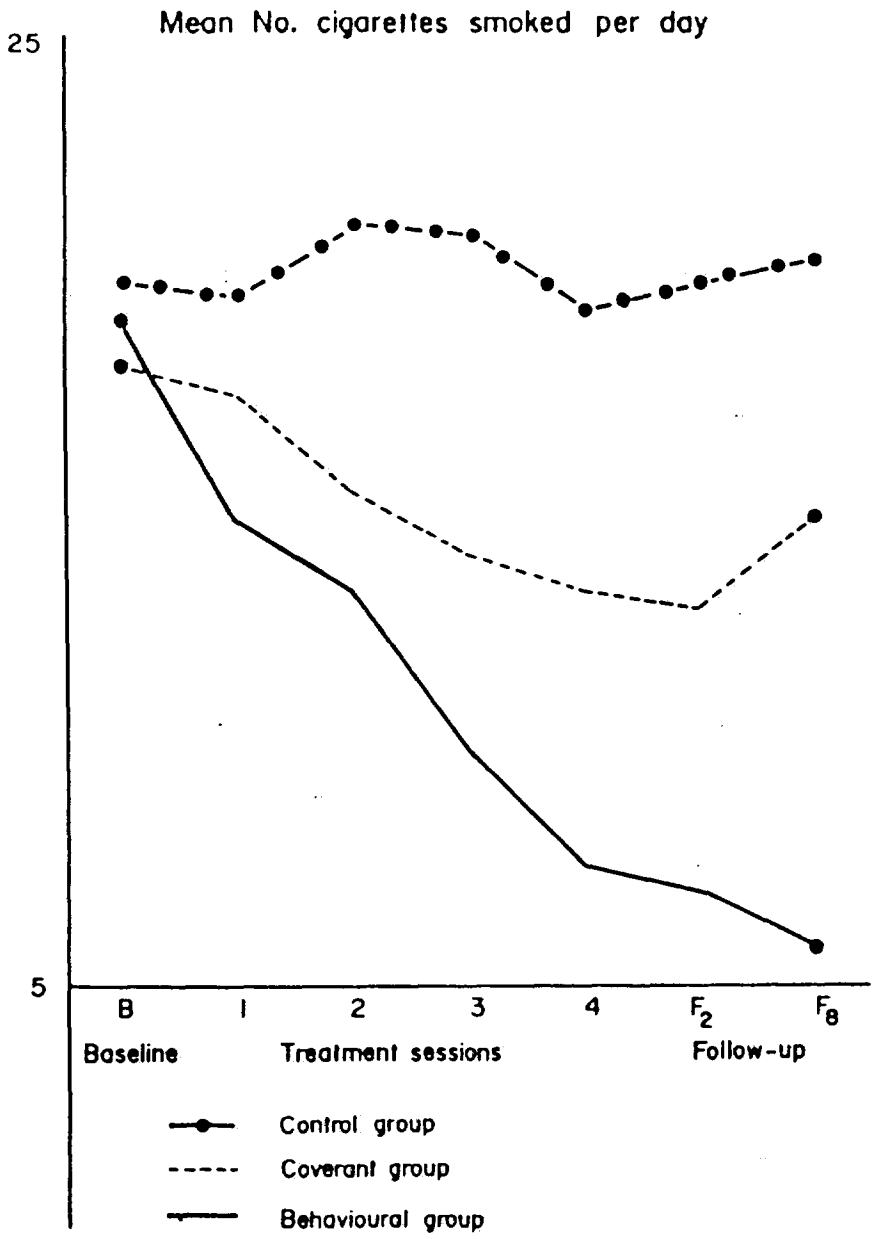
Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

FIG. 2.

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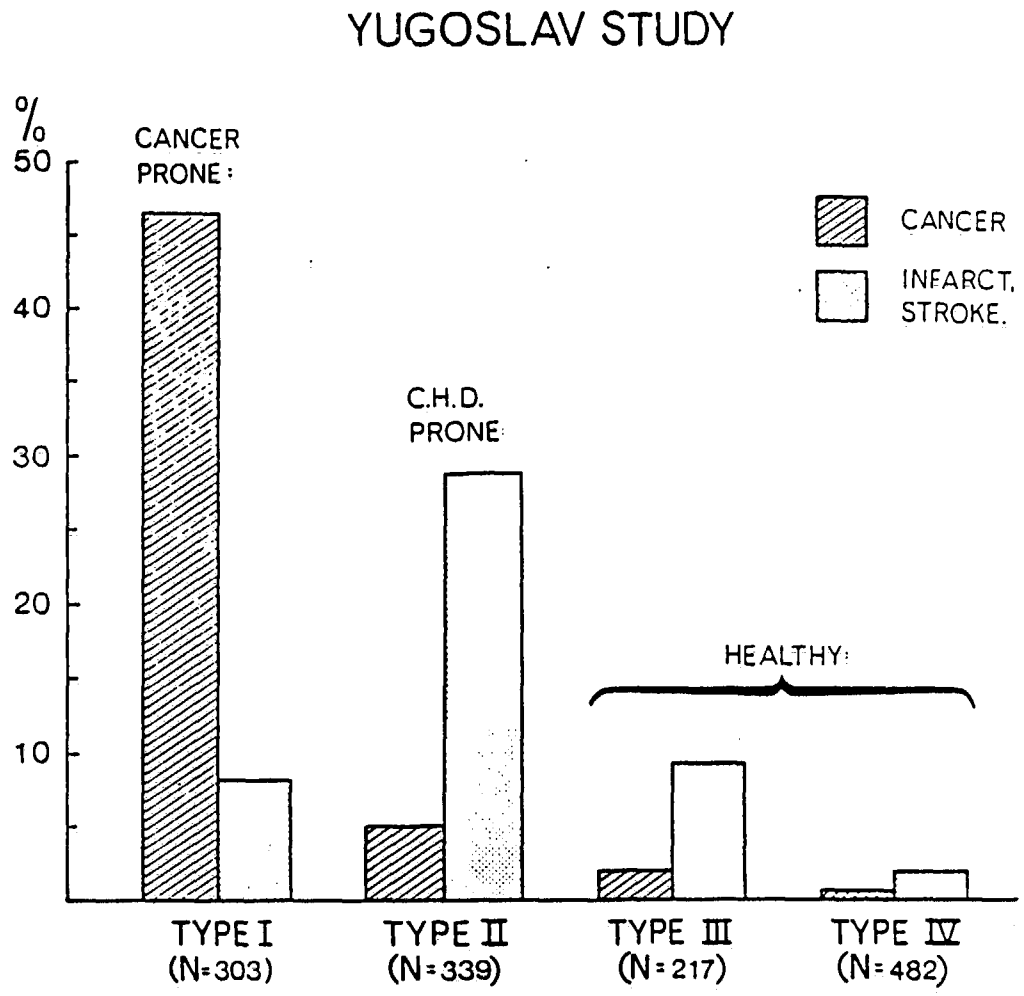
Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

FIG. 3.

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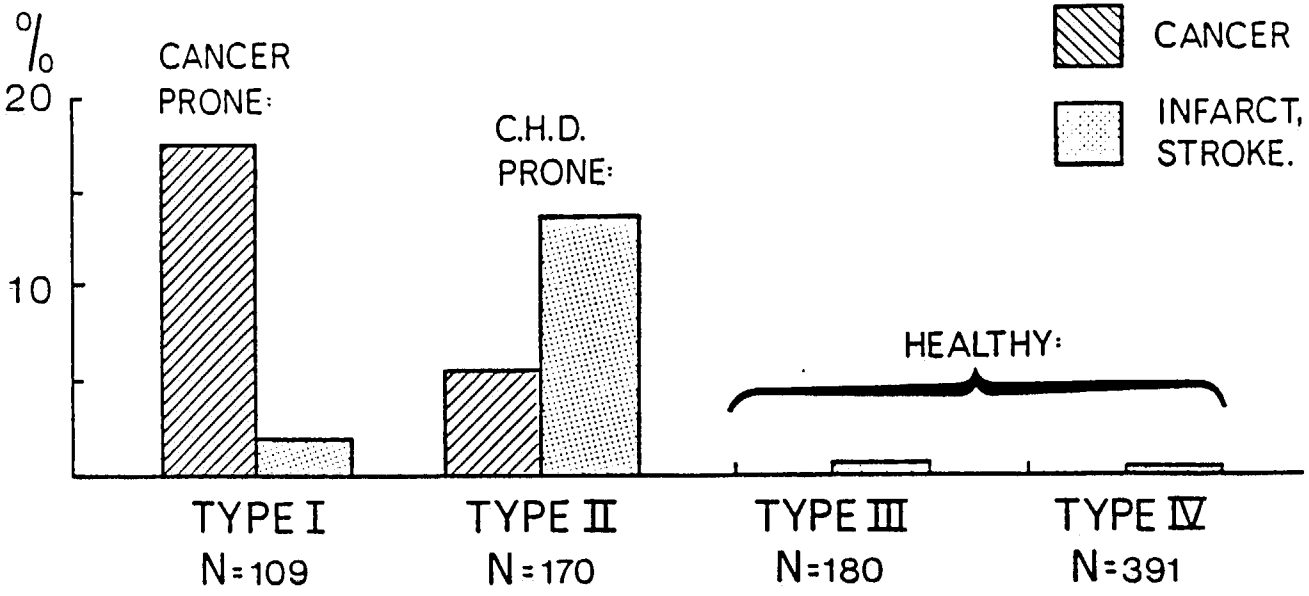
Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

FIG. 4.

# HEIDELBERG STUDY
## (normal group)

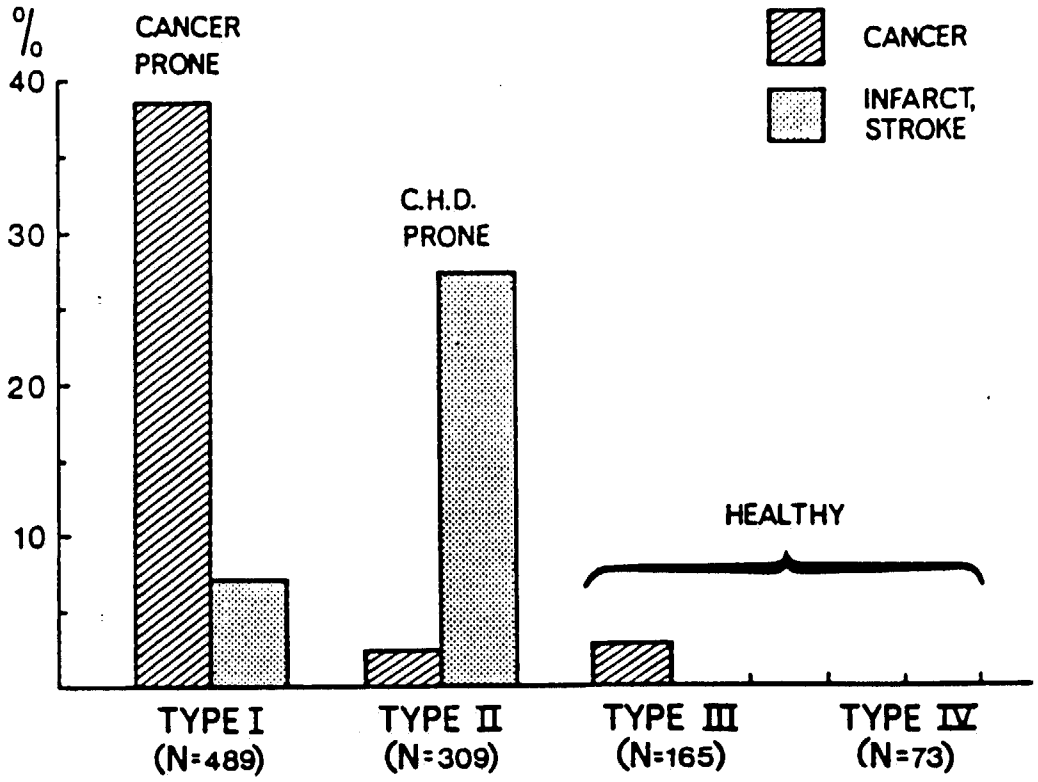



20250250240

Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

Fig. 5.

# HEIDELBERG STUDY
## (stressed group)

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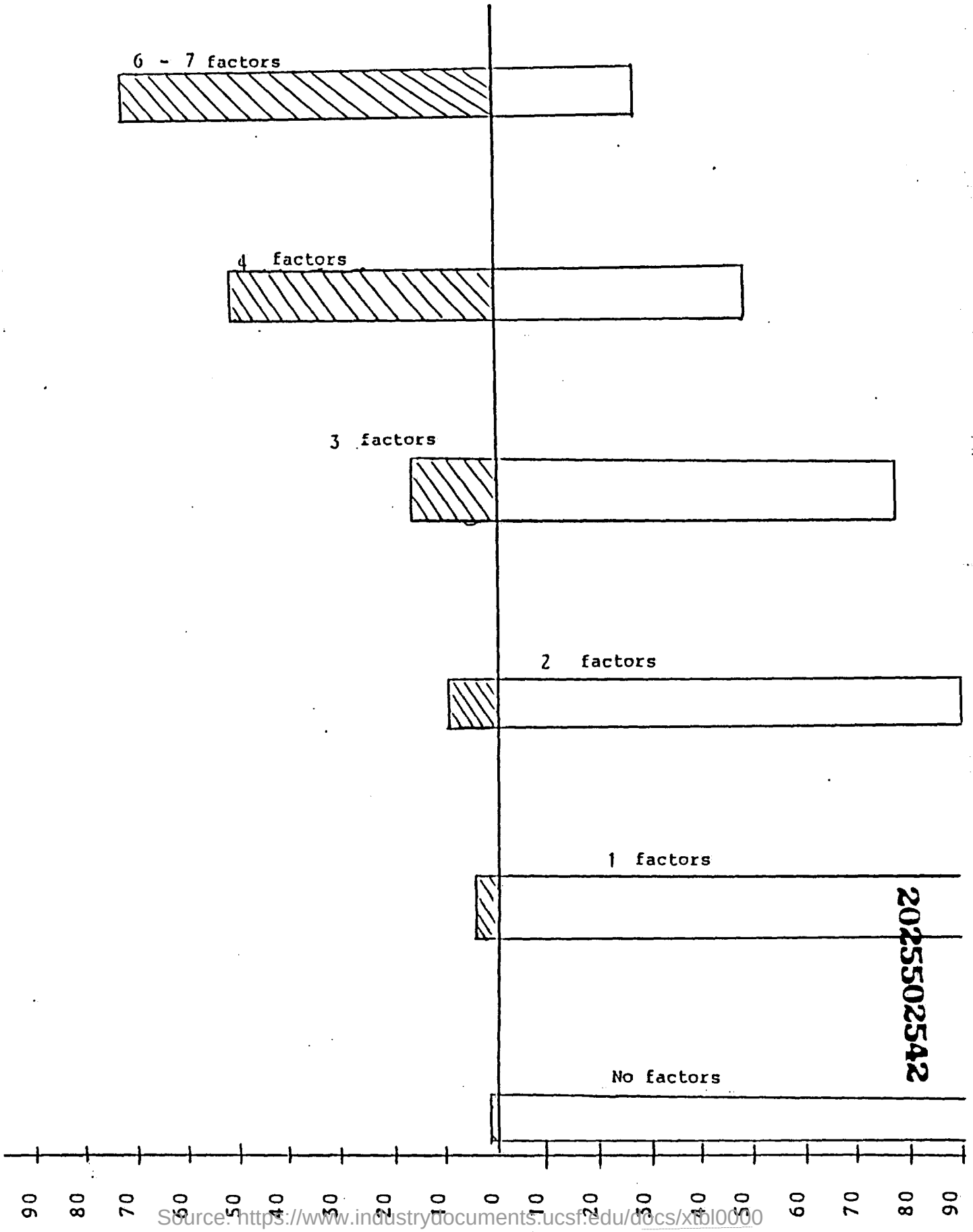
Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

Fig. 6.

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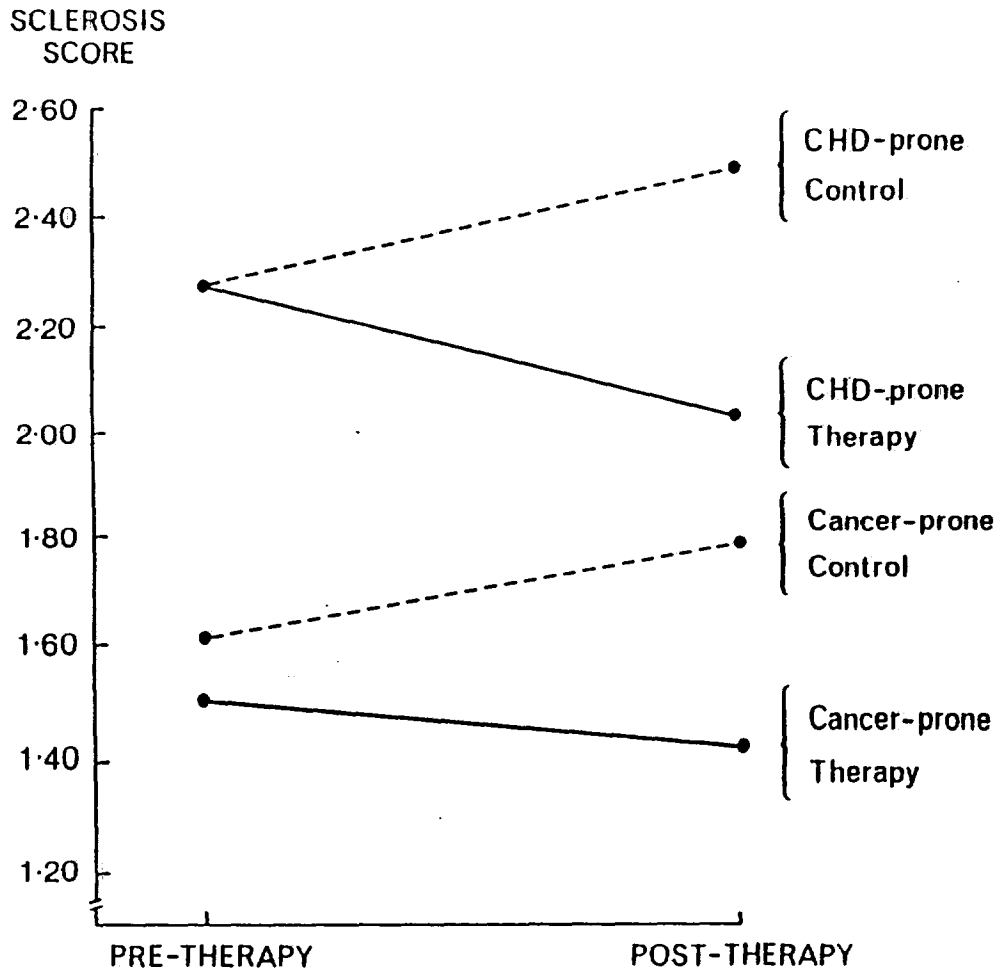
Source: https://www.industrydocuments.ucsf.edu/docs/xbl0000

FIG. 7.

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

APPENDIX

SHORT DISEASE-PRONENESS INVENTORY

...

(For scoring, see instructions at end of inventory)

1) I find it very difficult to stand up for myself. YES NO

2) I have been complaining for years about various unfavourable conditions, but am not able to change them. YES NO

3) I am mainly concerned with my own well-being. YES NO

4) I am usually content and happy with my daily activities. YES NO

5) I can express my feelings only when there are good reasons for them. YES NO

6) I don't believe in social rules, and don't pay much attention to other people's expectations or the obligations I may have towards them. YES NO

7) I cannot live happily and contentedly with nor without a particular person. YES NO

8) I prefer to agree with others, rather than assert my own views. YES NO

9) Certain people are the most important causes

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of my personal misfortunes. YES NO

10) I alternate to a great degree between the positive and negative evaluation of people and conditions. YES NO

11) When I cannot achieve closeness with someone who is emotionally important to me, I have no difficulties in letting them go. YES NO

12) I have difficulties in showing my emotions because for every positive emotion there is a negative one. YES NO

13) My behaviour towards other people alters from being very friendly and good-natured to being very hostile and aggressive. YES NO

14) I cannot live happily and contentedly in the presence or the absence of certain states or conditions; e.g. I need my work, but am unhappy doing it. YES NO

15) I tend to act more to fulfill the expectations of people close to me, rather than look after my own needs. YES NO

16) Certain conditions or situations are the most important causes of my personal misfortunes. YES NO

17) With people I love, I keep changing from keeping them at a great distance to stifling dependence, and from stifling dependence to

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

excessive distancing. YES NO

18) I can usually arrange things so that people who are emotionally important to me are as close to or as distant from me as I wish. YES NO

19) Reason, rather than emotion, guides my behaviour. YES NO

20) I often expect others to fulfill agreements very strictly, but do not believe in doing so myself. YES NO

21) I often have thoughts which terrify me and make me unhappy. YES NO

22) I tend to give in and abandon my own aims in order to achieve harmony with other people. YES NO

23) I feel helpless against people or conditions which cause great unhappiness for me, because I cannot change them. YES NO

24) When I am in a situation which I experience as threatening, I immediately try to get other people to help and support me. YES NO

25) When I fail to achieve my objectives, I can easily change tack. YES NO

26) When people make emotional demands on me, I usually react only rationally, never emotionally. YES NO

27) I usually act in a spontaneous manner, following my immediate feelings without

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

considering the actual consequences. YES NO

28) Relations with certain people are always pretty unsatisfactory, but there is nothing I can do about it. YES NO

29) I am unable to express my feelings and needs openly to other people. YES NO

30) I always seem to be confronted with the undesirable aspects of people and conditions. YES NO

31) When someone who is emotionally important to me hurts me ever so slightly, I immediately dissociate myself from that person. YES NO

32) I can manage to live fairly contentedly with or without someone who is emotionally important to me. YES NO

33) I am quite unable to allow myself to be guided by emotional considerations. YES NO

34) I often feel like attacking other people and crushing them. YES NO

35) Certain situations and states (e.g. at my place of work) tend to make me unhappy, but there is nothing I can do to alter things. YES NO

36) I tend to accept conditions which work against my personal interests without being able to protest. YES NO

37) Certain people keep interfering with my

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

personal development. YES NO

38) I expect others to live up to the highest moral standards, but do not feel that these are binding on myself. YES NO

39) I can usually change my behaviour to suit conditions. YES NO

40) My actions are never influenced by emotions to the degree that they might appear irrational. YES NO

41) When my partner demonstrates love towards me, I sometimes become particularly aggressive. YES NO

42) Certain bodily conditions (e.g. being overweight) make me unhappy, but I feel unable to do anything about it. YES NO

43) I often feel inhibited when it comes to openly showing negative feelings such as hatred, aggression, or anger. YES NO

44) Certain conditions keep interfering with my personal development. YES NO

45) I seek satisfaction of my own needs and desires first, regardless of the needs and rights of others. YES NO

46) I am usually capable of finding new points of view and successful, sometimes surprising,

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

solutions for problems. YES NO

47) I always try to do what is rational and logically correct. YES NO

48) When I feel like attacking someone physically, I have no inhibitions about doing this at all. YES NO

49) I can relax bodily and mentally only very rarely; most of the time I am very tense. YES NO

50) I am inclined not to be demonstrative when emotional shocks upset me. YES NO

51) I cannot control excitement or stress in my life because this is dependent on the actions of other people. YES NO

52) When I make emotional demands on another person, I require immediate satisfaction. YES NO

53) I am independent in what I do, and do not depend on other people when this works to my disadvantage. YES NO

54) I always try to express my needs and desires in a rational and reasonable manner. YES NO

55) I have no inhibitions in hurting myself physically if I feel like doing so. YES NO

56) I have great difficulties in entering into happy and contended relations with people. YES NO

57) When I feel emotionally let down I tend to be

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

paralysed and inhibited. YES NO

58) I cannot control excitement or stress in my life because this depends on conditions over which I have no control. YES NO

59) I usually find fulfilment in everyday situations which are <u>not</u> subject to ordinary rules, regulations and expectations. YES NO

60) When things don't work out, this does not make me give up, but rather makes me change my way of doing things. YES NO

61) I try to solve my problems in the light of relevant and rational consideration. YES NO

62) I resent all moral obligations because they hamper and inhibit me. YES NO

63) I am helpless when confronted with emotional shocks, depression or anxiety. YES NO

64) When something terrible happens to me, such as the death of a loved one, I am quite unable to express my emotions and desires. YES NO

65) I can express my aims and desires clearly but feel that it is quite impossible to achieve them. YES NO

66) As soon as someone becomes emotionally

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

important for me, I tend to place contradictory demands upon them, such as "Don't ever leave me" or "Get away from me." YES NO

67) When things lead to harmful results for me, I have no trouble in changing my behaviour to make for success. YES NO

68) I only believe in things which can be proved scientifically and logically. YES NO

69) When it benefits me I have no hesitation in lying and pretending. YES NO

70) I am seldom able to feel enthusiasm for anything. YES NO

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000

## Scoring Instructions for Short Disease-Proneness Inventory:

Type 1: Add "YES" answers to questions:

1, 8, 15, 22, 29, 36, 43, 50, 57, 64.

Type 2 Add "YES" answers to questions:

2, 9, 16, 23, 30, 37, 44, 51, 58, 65.

Type 3: Add "YES" answers to questions:

3, 10, 17, 24, 31, 38, 45, 52, 59, 66.

Type 4a: Add "YES" answers to questions:

4, 11, 18, 25, 32, 39, 46, 53, 60, 67.

Type 4b: Add "NO" answers to questions:

7, 14, 21, 28, 35, 42, 49, 56, 63, 70.

Type 5: Add "YES" answers to questions:

5, 12, 19, 26, 33, 40, 47, 54, 61, 68.

Type 6: Add "YES" answers to questions:

6, 13, 20, 27, 34, 41, 48, 55, 62, 69.

To decide on TYPE, add 4a and 4b, and divide by 2.
A person's type is then the one on which he has the highest score. This is the ipsative way of scoring.

Another method is to take all 6 scores into account:

Type 1 is the cancer-prone type;
" 2 " " coronary heart disease-type;
" 3 " " alternating reaction type - reasonably healthy;
" 4 " " autonomous, healthy type;
" 5 " " rational-antiemotional type; and
" 6 " " anti-social, egocentric type.

(For further details, see Grossarth-Maticek & Eysenck (1990).

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Source: https://www.industrydocuments.ucsf.edu/docs/xtbl0000